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MOTOR EXAMINATIONS IN PSYCHIATRY

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In this series, Drs. Sanders and Gillig explain how aspects of the neurological examination can aid in differential diagnosis of some common (and some uncommon) disorders seen in psychiatric practice.

ABSTRACT

The capacity for voluntary motor activity underpins all behavior. Although psychiatrists are acutely aware of behavior, we tend to think of its abstract motives more than its concrete mechanisms. This article reviews the basic brain mechanisms of voluntary motor activity, the most useful pyramidal tract or upper motor neuron signs, and their relevance to specific patient groups of interest to psychiatrists.

INTRODUCTION

Although psychiatrists are concerned with internal mental activities, psychiatric assessment is based almost exclusively on behavior involving the voluntary motor system, including speech. Because assessment of the mind is inescapably linked to assessment of motor behavior, psychiatric diagnosis has become more explicitly behavioral in recent decades.

Perhaps it should be no surprise then that psychiatric conditions more often involve disturbances of motor planning and performance

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than of other neurological or neuropsychological domains of functioning, and that psychiatric and movement disorders are highly comorbid. Psychiatric medications are prone to adversely affect motor functioning. Also, a minor change in motor activity can be the only indication of a shift in emotional state.

Because motor activity is so important to understanding their patients, psychiatrists become alert to it. However, failure to recognize motor disorders and limitations can gravely limit clinicians' ability to make sense of their observations. Common examples include the following: interpreting akathisia as anxiety, hypokinesis as depression, and mutism as anger. Further, motor signs and symptoms are sometimes dismissed prematurely as "functional" (i.e., without physiological basis) in psychiatric patients.

In this article, we will review the basic anatomy and physiology of the voluntary motor system, motor signs and symptoms in major psychiatric conditions and in important comorbid conditions, and efficient methods for assessing motor function, including strength, motor sequencing, and basic tests of motor response selection and control. We have previously discussed cranial nerves, gait, and cerebellar functions. The extrapyramidal motor system, reflexes, and "functional" signs will be covered in future entries.

RELEVANT ANATOMY AND PHYSIOLOGY

The traditional centerpiece of voluntary motor activity is the corticospinal (pyramidal) motor system. The first neuron originates in the precentral gyrus (i.e., motor strip), the premotor area, the supplementary motor area, or even the postcentral gyrus. The axon projects through the internal capsule, crosses over (mostly at the medullary "decussation of the pyramids"), proceeds down the spinal cord (mostly in the lateral corticospinal tract), and synapses at the anterior horn at the spinal level of exit. The second neuron, the "alpha motor neuron," proceeds from the anterior horn to the skeletal muscle, where it activates extrafusal muscle fibers. A *motor unit* is an alpha motor neuron and the extrafusal muscle fibers that it innervates. Rather than controlling all voluntary motor activity as was once believed, the corticospinal tract mostly controls distal limb muscles, particularly flexor muscles.

The corticoreticular/reticulospinal system accounts for the axial and extensor musculature, although it is not as cleanly wired. Corticoreticular axons proceed from cortical centers as do corticospinal axons, but divert to the brainstem reticular formation, where they synapse in crossed and uncrossed fashion. The pontine, medial part of the reticular formation project reticulospinal neurons that primarily activate alpha motor neurons of the axial and proximal limb muscles and particularly extensor muscles.

The corticorubral/rubrospinal system is similar to the corticospinal, the chief distinction being a relay in the midbrain red nucleus. As the red nucleus receives input from the thalamus and cerebellum, these structures are given this opportunity to influence voluntary motor signals.

The neuron or neurons above the anterior horn neuron are termed *upper motor neurons* (UMN), and lesions affecting them behave differently from the generally peripheral nervous system lesions affecting lower motor neurons (LMN). After a brief period, the UMN lesion will leave the affected area hypertonic, spastic, and hyperreflexic, whereas the LMN lesion will leave the affected area hypotonic, flaccid, and hyporeflexic. Both UMN and LMN lesions will leave the area weak and ultimately atrophic.

Several similar but distinct circuits help control motor planning and control. They start in the frontal cortex then project to the basal ganglia, which processes this input via two pathways (a monosynaptic direct and a polysynaptic indirect pathway) before projecting both to the thalamus, which feeds back to cortex and basal ganglia and to brainstem/spinal cord. Both the direct and indirect pathways are influenced by the famous dopaminergic nigrostriatal tract. The balance between direct and indirect pathways, regulated by dopamine circuits, is an important determinant of extrapyramidal motor function.[1]

Similar circuits are involved in decision-making and behavioral reinforcement. Information about reward-related learning flows from the prefrontal cortex to the striatum, which also interacts with sensorimotor cortex. The striatum projects to substantia nigra (pars reticulata, as opposed to the pars compacta of nigrostriatal fames) and the globus pallidus, which projects to thethalamus, which projects to the cortex. Reward and reward prediction strongly influence goal-directed and habitual behavior by way of separate circuits from prefrontal cortex to ventral striatum and amygdala. Dopamine from the substantia nigra (i.e., pars compacta) and the ventral tegmentum influences both the dorsal striatum, where it affects motor execution, and the ventral striatum, where it affects motor/behavioral choices.[2]

The basal ganglia consist of a group of structures given confusing terms, which bear reviewing. The *neostriatum*, often called *striatum*, consists of the caudate and putamen; the internal capsule looks like a stripe as courses through the combined structure, hence the term. The third major component of the basal ganglia, the globus pallidus, is also called the paleostriatum. The global pallidus and putamen, which lie together ventral to the internal capsule, are sometimes referred to jointly as the lenticular nucleus. Corpus striatum refers to the three nuclei together. The subthalamic nucleus is another important part of the basal ganglia system. The midbrain substantia nigra pars compacta, source of dopamine

neurons, is essentially part of this system as well. These structures are highly connected with frontal and also parietal cortex. The ventral striatum, consisting of nucleus accumbens and olfactory tubercle, is not part of this motor system, but is connected with primarily limbic structures.

SIGNS AND SYMPTOMS

Pyramidal tract signs are not strongly associated with most psychiatric illness, but are important in assessment of psychiatric patients. These signs point to brain disease, which could, of course, be responsible for the psychiatric syndrome. This is most often the case with dementia, which has a cerebrovascular etiology in a significant minority of cases.[3]

When it comes to identifying cerebral lesions in patients without obvious deficits, the most sensitive neurological signs (all have good specificity) are upper extremity motor tests.[4,5] These include the digiti quinti sign, finger rolling test, the forearm rolling test, strength testing by confrontation, rapid alternating movements (diadochokinesis and fist opening/closing), pronator drift, and unilaterally diminished arm swing.

For all the untold numbers of neurologic examinations that have been conducted on psychiatric patients, there is strikingly little published data on the results. The more routine the test (e.g., tests for focal weakness), the more barren the literature.

Pyramidal tract, or localizing motor signs, are not elevated as a group in studies of schizophrenia when compared with healthy controls. Some studies have found high rates of drift in schizophrenia,[7,8] but it is possible that these results reflect impersistence. Studies find comparable grip strength in schizophrenia and healthy comparison groups.[9–11] Not surprisingly, patients with schizophrenia had less abnormal lateralization of grip strength than a group of "organic" patients.[12] Grip strength is more precisely measured than drift and may be less vulnerable to bias, but it also tasks more distal muscles than drift. Proximal muscle weakness is considered a sign of myopathy, and there is evidence supporting a subtle myopathy in schizophrenia.[13] However, drift is vulnerable to confounds, such as a painful shoulder, and drift with normal neuroimaging and without other localizing signs is not rare. The question of proximal versus distal weakness in schizophrenia could be readily settled empirically. Obviously, weakness is vulnerable to deconditioning, so it would be best approached with patients in prodrome or early illness. In the mood and anxiety disorders, focal motor weakness has not been observed. In late-onset depression, and in the related "vascular depression," focal weakness and other focal signs are elevated.[14]

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There are myriad other tests of higher-order motor functioning. Some of the most basic motor tests in neuropsychology are tapping speed, simple reaction time, and grip strength. In usual neuropsychiatric testing, the most common tests are of praxis, motor sequencing, inhibition of automatic responses, and persistence.

The term *apraxia* is about 140 years old and refers to the inability to organize simple instrumental actions, in spite of intact basic motor capabilities. Although one might hear of numerous types of apraxia, for the past 90 years the following three types have persisted: 1) ideomotor apraxia (temporal or spatial errors), 2) ideational or conceptual apraxia (content errors), and 3) limb-kinetic apraxia (slow, clumsy movements). Apraxia can be assessed with any body part, with real or imaginary props or tools, with strictly verbal instruction or with demonstration as to what activity is expected. Apraxia can be attributed to a variety of brain structures, including left hemisphere, the frontal cortex, basal ganglia, and parietal cortex.[15–17]

Motor sequencing tests were developed by Luria[18] to test prefrontal integrity, mostly based on studies of adults who had experienced injuries to the frontal convexity (i.e., dorsolateral prefrontal) after developing normally. These include the fist-ring, the fist-edge-palm, and the alternating fist-palm tests. Functional imaging of intact persons does not clearly confirm that these are prefrontal tasks.[19–21] These motor sequencing tests were incorporated in neuropsychological[22] and neurological[23–25] batteries, and have been administered to large numbers of psychiatric patients, particularly patients with schizophrenia, bipolar disorder, and obsessive compulsive disorder (OCD). Motor sequencing can be quite severely impaired in primary psychosis and dementia and also in bipolar disorder and OCD. These deficits are consistent and striking in the psychotic disorders, but it is unresolved whether they are more impaired in schizophrenia than in other major mental disorders.[26–29]

Motor inhibition deficits, indicated by inappropriate responding to nontarget stimuli, are also common in the primary psychotic disorders. A common example is the Go-No Go test, which has been often studied in schizophrenia.[30–33] Similarly, impersistence, indicated by failure to maintain an unnatural position past a few seconds, is represented in some "soft sign" batteries.[7,25]

EXAMINATION METHODS

Strength testing with attention to symmetry is useful and important. Observation often reveals asymmetry of muscle tone and strength. For example, spontaneous gait may reveal circumduction (i.e., a swinging

around of one leg rather than stepping directly forward), suggesting weakness in the hip flexor.

Confrontational strength testing usually consists of bilateral comparisons. It is only sensitive with the weaker muscle groups, because stronger muscle groups (e.g., proximal legs and hips) can still overpower the examiner's testing arms on examination after considerable loss of strength.

We will review some of the less publicized signs that are also among the most sensitive in detecting subtle brain lesions.[4,5]

The digiti quinti sign is quite simple. The clinician asks the patient to extend the arms and fingers with the palms down. With mild weakness, the fifth finger on the weak side assumes a slight abduction, creating a visible space between it and the ring finger.[34]

The finger rolling test and forearm rolling test are similar, although the former may be more sensitive. In the forearm rolling test, each forearm is rapidly rotated around the other for five seconds in each direction. If one forearm orbits around the other (the arms don't describe a similar sized circle), the less active arm is paretic. The finger rolling test is similar to the forearm rolling test except with each index finger rotated around the other for five seconds in each direction.[35]

Pronator drift is fairly well known but worth reviewing. The clinician asks the patient to extend the arms and fingers with the palms up and hold the position for 10 to 20 seconds with his or her eyes closed. Two upper motor neuron signs, often in combination, may be seen: pronation (wrist rotates inward on affected side) and drift (arm drifts downward on affected side).

Grip strength is casually assessed by asking the patient to squeeze the clinician's fingers. Mechanical and electromechanical grip strength devices yield specific measures, which can be compared to norms; variability in performance can be used to evaluate effort/deception.[36,37]

In the most common tests of praxis, which can be used to simultaneously assess handedness, the clinician asks the patient to demonstrate common tasks (e.g. hammering a nail).

Two of the more common motor sequencing tests are the fist-ring test and the fist-edge-palm test. In the fist-ring test, the patient alternates placing a hand on a table in a fist and in a ring. In the fist-edge-palm test, the patient strikes the fist, then (medial) edge of the hand, then the palm of one hand on the table surface. In both of these tasks, the sequence is repeated several times. The tasks are introduced with verbal description and demonstration.

The popular response suppression task is the Go–No Go. This is one of its more common variations: the clinician holds up 1 or 2 fingers at a time, for 1 to 2 seconds, waiting 1 to 2 seconds between stimuli. The clinician then asks the patient to signal one finger is held up and to do nothing when two fingers are held up. Errors of commission (e.g., responding to 2 fingers) reflect failures of response suppression.

Impersistence tasks can usually be incorporated in other tests, such as the test for motor drift and the Romberg. We have previously mentioned the Romberg, but not described it. While demonstrating, tell the patient to stand with feet completely together, fingers spread, and arms fully abducted and parallel to the floor. Finally, ask the subject to close the eyes and hold the pose until stopped. The Romberg is commonly used to test for balance and position sense, but it can often serve also as a test for impersistence. Simply note whether or not the patient maintains the pose for 30 seconds (without encouragement). Impersistence consists of failing to continue with the task (e.g., opening the eyes or returning to a relaxed position without prompting).

SUMMARY

Understanding human behavior sometimes requires an understanding of the individual's motor capabilities. We summarized the basics of the human corticospinal tract for control of voluntary motor activity and some of the motor association centers, which help to determine and control motor behavior. Corticospinal tract signs are uncommon in psychiatric disorders other than dementia, in which they suggest focal brain disease (e.g., cerebrovascular disease). The most useful motor signs for detecting subtle brain lesions are the relatively unknown digiti quinti sign, the finger rolling test, and the forearm rolling test. Fortunately these tests are quick and easy for both patient and doctor.

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